(5'CCGAATTCCATATGAGCAACGAT AATGAC 3' (SEQ. ID. No. 40)) and bottom primer with *Xba*I cutting site (5'CCTCTA GAGGATNNCTAGCTGAGCTTGCCGAGG 3' (SEQ. ID. No. 41)) were synthesized and used for PCR amplification of *hemAT-Hs* gene. The PCR amplicon was cloned into TOPO cloning vector (Invitrogen) and transformed into *E. coli* competent cells. The plasmid containing *hemAT-Hs* gene in TOPO vector was subcloned into pKJ427 vector by *NdeI/Xba*I double digestion. The *hemAT-Hs*/pKJ427 construction was confirmed by PCR as well as *NdeI/Xba*I double digestion and transformed into Δ*htrVIII* strain using standard halobacteria transformation protocol. Individual colonies were checked by PCR and immunoblot to confirm the expression level of HemAT-*Hs*; Construction of OI3428: A 322 bp fragment interior to HemAT-*Bs* was amplified from the *B. subtilis* wild type strain OI1085 chromosome using primers with overhanging *Hind*III and *Bam*H1 sites (reverse primer: 5' TATGGGATCCCTTGTTCATCACGGGTCTNTTGG 3' (SEQ. ID. No. 42), forward primer: 5' GATAAAGCTTGATCATAGCTCAGTTGACCG 3' (SEQ. ID. No. 43)). This PCR fragment was digested with *Hind*III and *Bam*H1 and cloned in the integration vector pHV501 (Vagner et al., Microbiology, 144(Pt 11):3097-3104 (1998)) to create pMK1. The resultant plasmid pMK1 was transformed into OI1085 and HemAT-*Bs* mutants were selected by erythromycin resistance. Integration of the pMKI into the correct locus was checked by linkage analysis. The *hemAT-Bs* locus is 30% linked to the *glyk* locus as determined from the *B. subtilis* chromosomal map. GLY+ transductants were selected and scored for erythromycin resistance. Construction of OI3498: The entire HemAT-*Bs* gene including the native promoter and the ribosome binding site was amplified from the *B. subtilis* wild type strain OI1085 chromosome using primers with overhanging *EcoR*I and *BamH*I sites (HemAT-*Bs* amyup: 5' TGCTGAATTCGCAGCTTTCATTCATGTTTCCC 3') (SEQ. ID. No. 44), HemAT-*Bs* amydown: 5' TTAGGGATCCGTCAACTGATTTTTAA TTTAAGTTAC 3') (SEQ. ID. No. 45)). The PCR amplicon was digested with *EcoRI/BamH*I and cloned into the amyE integration vector pDG1730 (Guerout-Fleury et al., Gene, 180(1-2):57-61 (1996), which is hereby incorporated by reference) to produce pKZ2. The resultant plasmid pKZ2 was digested with *BglI/Xba*I to ensure a double crossover event into the *amyE* locus and then transformed into OI3428 to select for Spec-R. HemAT-*Bs* overexpression R4: Overexpression construction in *E. coli*: The HemAT-*Bs* overexpression construction was performed as follows: *B. subtilis* OI1085 genomic DNA was used for the PCR amplification of HemAT-*Bs* gene by Pfu DNA polymerase using two primers (Top primer with *BamH*I restriction site: 5'ATATGGATCCAAGGGGGATCATTGTAATGTTA

TTTAAAAAAG 3' (SEQ. ID. No. 46), Bottom primer with *Pst*I site: 5' ATTACTGCAGCA ACTGATTTTTAATTTAAGTTT ACATAATGAACGC 3' (SEQ. ID. No. 47)). The PCR amplicon was cloned into TOPO cloning vector (Invitrogen) and transformed into TOP 10 *E. coli* competent cells. Colonies were tested for the presence of plasmids containing the correct insert. The recombinant plasmid was digested with *BamH*I and *Pst*I and the insert with HemAT-*Bs* open reading frame was cloned into the pMALcII expression vector (New England Biolabs, Inc).

Please replace Table 3, appearing at the top of page 28, with the following:

Table 3. Names and sequences (5' to 3') of primers used in HemAT-*Hs* truncation.

Primer Name	**Sequence (5' to 3')**	
*hemAT-Hs EcoR*I/*Nde*I top	ccgaattccatatgagcaacgataatgac	SEQ. ID. No. 48
hemAT-Hs 151 *BamH*I/*Xba*I bot	ctctagaggatccctagtcgtcggcaagcgcgtcc	SEQ. ID. No. 49
hemAT-Hs 250 B/X bot	cctctagaggatccntagacgtcagccatgcggtc	SEQ. ID. No. 50
hemAT-Hs 230 B/X bot	cctctagaggatccctaggcgacgtcctgcgaggtcgcc	SEQ. ID. No. 51
hemAT-Hs 210 B/X bot	cctctagaggatccctacgcgttcgccaactcctggcggc	SEQ. ID. No. 52
hemAT-Hs 190 B/X bot	cctctagaggatccctagatgtaggtgtccattgcgatc	SEQ. ID. No. 53
hemAT-Hs 170 B/X bot	cctctagaggatccctaccgggccacgagttcgtcgac	SEQ. ID. No. 54
hemAT-Hs 205 B/X bot	cctctagaggatccctactggcggctgtcgatctcgtc	SEQ. ID. No. 55
hemAT-Hs 200 B/X bot	cctctagaggatccctactcgtcgtggaggcgctgggc	SEQ. ID. No. 56
hemAT-Hs 195 B/X bot	cctctagaggatccctactgggcgtacgagtcgatgtag	SEQ. ID. No. 57
hemAT-Hs 194 B/X bot	cctctagaggatccctaggcgtacgagtcgatgtaggtgtcc	SEQ. ID. No. 58
hemAT-Hs 193 B/X bot	cctctagaggatccctagtacgagtcgatgtaggtgtcc	SEQ. ID. No. 59
hemAT-Hs 192 B/X bot	cctctagaggatccctacgagtcgatgtaggtgtccattgcg	SEQ. ID. No. 60
hemAT-Hs 191 B/X bot	cctctagaggatccctagtcgatgtaggtgtccattgcg	SEQ. ID. No. 61

In the Claims:

Please cancel the second appearing claim 7 on page 39, lines 28-29.

Please cancel the second appearing claim 8 on page 39, lines 31-32.

Please add new claims 62 and 63 as follows:

64 ~~62.~~ (New) A fragment of the isolated heme-binding protein according to claim 1, wherein said fragment comprises a heme-binding domain.

65 ~~63.~~ (New) The fragment according to claim 4, further comprising a heterologous signal transduction domain.